

Effects of awareness diffusion and self-initiated awareness behavior on epidemic spreading - an approach based on multiplex networks

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Abstract

In this paper, we study the interplay between the epidemic spreading and the diffusion of awareness in multiplex networks. In the model, an infectious disease can spread in one network representing the paths of epidemic spreading (contact network), leading to the diffusion of awareness in the other network (information network), and then the diffusion of awareness will cause individuals to take social distances, which in turn affects the epidemic spreading. As for the diffusion of awareness, we assume that, on the one hand, individuals can be informed by other aware neighbors in information network, on the other hand, the susceptible individuals can be *self-awareness* induced by the infected neighbors in the contact networks (local information) or mass media (global information). Through Markov chain approach and numerical computations, we find that the density of infected individuals and the epidemic threshold can be affected by the structures of the two networks and the effective transmission rate of the awareness. However, we prove that though the introduction of the *self-awareness* can lower the density of infection, which cannot increase the epidemic threshold no matter of the local information or global information. Our finding is remarkably different to many previous results—local information based behavioral response can alter the epidemic threshold.

Key words: Multiplex networks, Infectious diseases, Awareness diffusion, Epidemic threshold.

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1 Introduction

The outbreaks of diseases can involve the diffusion of information in regard to the diseases, including the risk of infection, rumors, fears and so on, which can stimulate individuals to take spontaneous behavioral responses to protect themselves, thereby bring profound impacts on the spreading of disease [1,2,3,4,5,6]. For example, recent outbreaks of the H1N1 flu, the bird flu, and the severe acute respiratory syndrome (SARS) have brought the reduction of going out and the plenty of people wearing face masks. For this reason, there has been an increasing focus on the development of formal models aimed at investigating the interplay of epidemic spreading and information-based behavioral responses[7,8,9,10]. Such as, based on the assumption that the probability of susceptible individual going to the alter state is proportional to the number of infected neighbors, Sahneh *et al.* extended the SIS (Susceptible-Infected-Susceptible) model to a Susceptible-Alter-Infected-Susceptible (SAIS) model [11,12], and they found that the way of behavioral response can enhance the epidemic threshold; Meloni *et al.* studied a meta-population model that incorporates several scenarios of self-initiated behavioral changes into the mobility patterns of individuals, and they found that such behavioral changes do not alter the epidemic threshold, but may produce a negative impact on disease, i.e., the density of infection is increased [13]; In Refs. [14,15,16], authors investigated the effects of the information-based behavioral responses on the epidemic dynamics by designing the transmission rate as a function of the local infected density or the global infected density.

Though the effects of information-based behavioral responses on the epidemic dynamics have been studied by many authors, most of works assumed the spreadings of information and epidemic are in the same network. As we know, with the development of technology, information can fast diffuse through many different channels, such as, the word of mouth, news media, online social networks, and so on. In view of this, recent well-studied multiplex network theory has been used to mimic the interplay of information or related awareness and the epidemic dynamics[17,18,19,20]. For instance, Sahneh *et al.* have shown that the information dissemination spread in another network can help boost the resilience of the agents' population against the spreading and found optimal information dissemination for different topologies [21]; Wang *et al.* investigated the interplay of the epidemic dynamics and the information dynamics in multiplex network based on the SIR (R-recovery) model, and focused on the two fundamental quantities underlying any spreading process: epidemic threshold and the final epidemic prevalence [22]; Granell *et al.* established an SIS-UAU model to investigate the competing effects of the spreading of awareness and the epidemic dynamics in multiplex with the transmission rate of awareness as well as the structure of information network [23]. More recently, they further generalized their model by reducing the probability of

infected individuals becoming awareness and including the effect of a mass broadcast of awareness (mass media) on the epidemic dynamics [24].

Existing works either assume that individuals are self-initiated [13,12], that is, they become aware because their neighbors are infected, or individuals can only be informed and become aware by other aware neighbors [22,23], the combine effects of the two factors have not been well studied. In reality, individuals can obtain the disease information through many ways. Inspired these factors, in the current work, we study the interplay between the diffusion of awareness by incorporating the *self-awareness* effects and the epidemic dynamics under the framework of multiplex network. In the model, an infectious disease first spreads among population represented by the contact network, and then the outbreak of the disease stimulates some people (infected or susceptible individuals) become aware of the risk of infection, and they take some protections to reduce the probability of infection. Meanwhile, unaware individuals can be informed by other aware individuals through the information network or become *self-awareness* induced by the infected neighbors in contact network or mass media. The finding indicates that the additional self-initiated awareness mechanism can reduce the density of infection, however, which can not alter the epidemic threshold. The results are verified by the Monte-Carlo simulations and the microscopic Markov chain approach (MMCA).

The layout of the paper is as follows: we introduce the model in Sec. 2. The simulation results and theoretical analysis are presented in Sec. 3. Finally, Conclusions and discussions are presented in Sec. 4. The results for the global information-based *self-initiated* awareness are given in Appendix.

2 Model

In this work, we generalize the model of Ref. [23]. In that model, a multiplex includes two layers, one is physical layer representing the spreading of epidemic (contact network), and the other is information layer on where the diffusion of the awareness evolves (information network). All nodes represent the same individuals in both layers, but the connectivity is different in each of them. In the contact layer, a Susceptible - Infected - Susceptible (SIS) model is used to mimic the epidemic dynamics. That is to say, a susceptible node can be infected by one infected neighbor with certain probability, and the infected node can return to susceptible state with probability μ . On the information layer, the dynamical process of awareness is assumed to be similar to the SIS model, that is, an unaware node (U) can be informed by an aware neighbor (A) with probability λ , and the aware node can loss awareness and back to unaware state with probability δ . The interplay of the two processes is modelled as follows: once an individual is infected, s/he will certainly become aware, that

is, the probability is $\sigma = 100\%$. In addition, to distinguish the protective behaviors between the aware individuals and unaware individuals, let β and $\beta^A = \gamma\beta$ (here $0 \leq \gamma < 1$. If $\gamma = 0$, the aware individuals are completely immune to the infection.) be the probabilities of unaware and aware susceptible nodes to get infected, respectively.

From the description of the model, one can find that, on the one hand, the authors assumed that the infected individuals will *automatically* become aware and are willing to inform the disease information. As we know, in many cases, infected individuals are unwilling to tell others since they can be discriminated or isolated by others once others know they are infected by one certain disease. So we make a progress and assume that infected individuals becoming aware with probability $0 \leq \sigma \leq 1$. On the other hand, in the model, individuals can *only* be informed by their neighbors through the information network, that is to say, one individual has no chance to become aware once their neighbors are unaware. However, as proposed in many previous works, individuals can become *self-awareness* once their friends are infected or they are informed by the mass media. Thus, we further generalize the model as: a susceptible individual can go to aware state by *self-initiated* response with probability κ when contacting one infected friend. Therefore, the probability of the susceptible individual becoming awareness increases with the number of infected neighbors in the contact network [12]. Note that, for the original model in Ref. [23], awareness cannot break out if the probability $\sigma = 0$, so the roles of awareness cannot be played, however, in our model, the awareness can diffuse among population even though $\sigma = 0$ since susceptible individuals can become awareness by their *self-initiated* responses. When $\kappa = 0.0$ and $\sigma = 1.0$, our model returns to the original model in Ref. [23].

According to this scheme, an individual can be in four different states: susceptible and unaware(SU), susceptible and aware(SA), infected and unaware(IU), infected and aware(IA). The flow diagram of the model is given in Fig. 1.

3 Main results

3.1 Theoretical analysis

Denoting a_{ij} and b_{ij} be the adjacency matrices that support the SIS and UAU processes, respectively. The probability of i in one of four states at time t is denoted by $p_i^{SU}(t)$, $p_i^{SA}(t)$, $p_i^{IU}(t)$ and $p_i^{IA}(t)$ respectively. Assuming the probability of susceptible (infect) node i *not* being *informed* by any neighbors is $\theta_i(t)$ ($r_i(t)$), and the probability of unaware (aware) susceptible node i *not* being *infected* is $q_i^U(t)$ ($q_i^A(t)$). They are described as [23]:

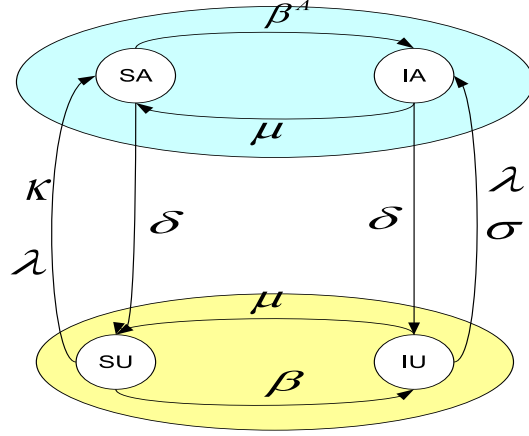


Fig. 1. Model description for the UAU-SIS dynamic. An individual can be in four different states: SU, SA, IU, and IA. The top (bottom) layer is the epidemic process for the aware (unaware) individuals, respectively. SU (SA) can be infected by an infectious neighbor in contact layer with a probability β ($\beta^A = \gamma\beta$). IU and IA recovers to SU and SA, respectively, with the same probability μ ; The left (right) flow is the awareness process for the susceptible (infected) individuals, respectively. SU can go to SA with a probability λ of being informed by an aware neighbor through information network, or induced by the infected neighbors in contact network with a probability κ . SA recovers to SU with a probability δ . IU can go to IA by informed the aware neighbors in information layer with a probability λ , or self-awareness with a probability σ . SA and IA can become unaware and return to SU and IU with the same probability δ .

$$\theta_i(t) = \prod_j (1 - b_{ji} p_j^A(t) \lambda) (1 - a_{ji} p_j^I(t) \kappa), \quad (1)$$

$$r_i(t) = \prod_j (1 - b_{ji} p_j^A(t) \lambda), \quad (2)$$

$$q_i^U(t) = \prod_j (1 - a_{ji} p_j^I(t) \beta), \quad (3)$$

$$q_i^A(t) = \prod_j (1 - a_{ji} p_j^I(t) \beta^A), \quad (4)$$

where $p_j^A(t) = p_j^{SA}(t) + p_j^{IA}(t)$ and $p_j^I(t) = p_j^{IU}(t) + p_j^{IA}(t)$ (Note: to simplify the model, we do not distinguish the infectivity of IA and IU, meanwhile, the diffusion capabilities of awareness for SA and IA are also the same.).

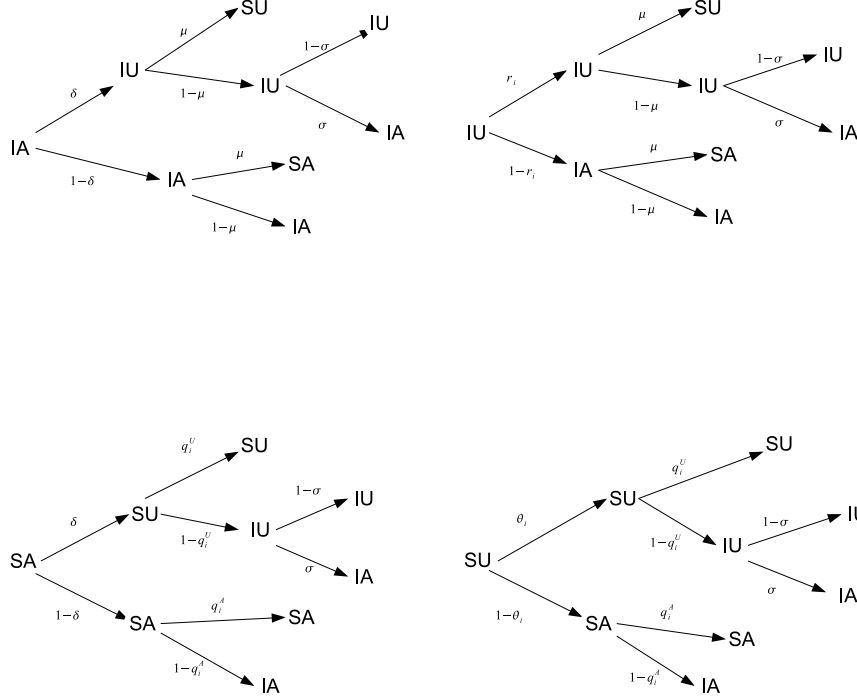


Fig. 2. Transition probability trees for the states of UAU-SIS dynamics in the multi-plex for per time step. The denotations of θ_i , r_i , q_i^U and q_i^A are given in Eqs. (1)-(4). Other parameters have the same denotations as in Fig. 1.

For each possible state at time t , which may give rise to four possible states at time $t + 1$ with certain probability, the transition probability trees for node i are illustrated in Fig. 2. According to the scheme in Fig. 2, one can easily write the Markov Chain Approach (MMCA) equations [25,26] for each state, for example, along the top branch of the four trees, one can read the probability $p_i^{SU}(t + 1)$ of SU at $t + 1$ as:

$$p_i^{SU}(t + 1) = p_i^{IA}(t)\delta\mu + p_i^{IU}(t)\gamma_i(t)\mu + p_i^{SA}(t)\delta q_i^U(t) + p_i^{SU}(t)\theta_i(t)q_i^U(t) \quad (5)$$

Similarly, the other three MMCA equations can be written as:

$$p_i^{SA}(t + 1) = p_i^{IA}(t)(1 - \delta)\mu + p_i^{IU}(t)(1 - r_i(t))\mu + p_i^{SA}(t)(1 - \delta)q_i^A(t) + p_i^{SU}(t)(1 - \theta_i(t))q_i^A(t), \quad (6)$$

$$p_i^{IU}(t + 1) = (1 - \sigma)\left\{p_i^{IA}(t)\delta(1 - \mu) + p_i^{IU}(t)r_i(t)(1 - \mu)\right\} \quad (7)$$

$$\begin{aligned}
& + p_i^{SA}(t)\delta(1 - q_i^U(t)) + p_i^{SU}(t)\theta_i(t)(1 - q_i^U(t)) \Big\}, \\
p_i^{IA}(t+1) = & \sigma \Big\{ p_i^{IA}(t)\delta(1 - \mu) + p_i^{IU}(t)r_i(t)(1 - \mu) + p_i^{SA}(t)\delta(1 - q_i^U(t)) \\
& + p_i^{SU}(t)\theta_i(t)(1 - q_i^U(t)) \Big\} + p_i^{IA}(t)(1 - \delta)(1 - \mu) \\
& + p_i^{IU}(t)(1 - r_i(t))(1 - \mu) + p_i^{SA}(t)(1 - \delta)(1 - q_i^A(t)) \\
& + p_i^{SU}(t)(1 - \theta_i(t))(1 - q_i^A(t)),
\end{aligned} \tag{8}$$

where $p_i^{SU}(t) + p_i^{SA}(t) + p_i^{IU}(t) + p_i^{IA}(t) \equiv 1$. When the system goes to the stationary state, we have $p_i^{SU}(t+1) = p_i^{SU}(t) = p_i^{SU}$ for SU state and equivalently for the SU, SA and IU states.

Since the epidemic threshold determines whether the epidemic can outbreak or die out, it is vital to analyze the effects of the different parameters on the epidemic threshold β_c . As stated in Ref. [23], near the threshold, the probability of nodes being infected is very low, i.e., $p_i^I = \varepsilon_i \ll 1$. Consequently, $q_i^A \approx 1 - \beta^A \sum_j (a_{ji}\varepsilon_j)$ and $q_i^U \approx 1 - \beta \sum_j (a_{ji}\varepsilon_j)$. Further approximate $q_i^A \approx 1$ and $q_i^U \approx 1$ by assuming $p_i^I = \varepsilon_i \rightarrow 0$, and then substitute both of them into Eq. (5) and Eq. (6), we obtain

$$p_i^{SU} = p_i^{SA}\delta + p_i^{SU}\theta_i \tag{9}$$

$$p_i^{SA} = p_i^{SA}(1 - \delta) + p_i^{SU}(1 - \theta_i) \tag{10}$$

Combing Eqs. (7–10), then a simple formula is obtained:

$$\mu\varepsilon_i = (p_i^{SU}\beta + p_i^{SA}\beta^A) \sum (a_{ji}\varepsilon_j). \tag{11}$$

With $\beta^A = \gamma\beta$, $p_i^U = p_i^{SU} + p_i^{IU} \approx p_i^{SU}$, $p_i^A = p_i^{SA} + p_i^{IA} \approx p_i^{SA}$ and $\varepsilon_i = p_j^{IU}(t) + p_j^{IA}(t) \ll 1$, then Eq. (11) can be rewritten as:

$$\sum [(1 - (1 - \gamma)p_i^A)a_{ji} - \frac{\mu}{\beta}\delta_{ji}]\varepsilon_j = 0, \tag{12}$$

here $\delta_{ji} = 1$ if $i = j$; otherwise, $\delta_{ij} = 0$.

Defining matrix H with elements:

$$h_{ji} = (1 - (1 - \gamma)p_i^A)a_{ji}, \tag{13}$$

Eq. (12) can be read as

$$H\varepsilon = \frac{\mu}{\beta}\varepsilon, \tag{14}$$

where $\boldsymbol{\varepsilon} = (\varepsilon_1, \varepsilon_2, \dots, \varepsilon_N)^T$ with T be the vector transportation.

The non-trivial solutions of Eq. (14) are eigenvectors of H , whose eigenvalues are equal to μ/β . Therefore, the onset of the epidemics is given by the largest eigenvalue of H , *i.e.*, $\Lambda_{max}(H)$,

$$\beta_c = \frac{\mu}{\Lambda_{max}(H)}. \quad (15)$$

3.2 Numerical simulations

To verify our theoretical results, as in Ref. [23], we build a configuration network with degree distribution $P(k) \sim k^{-2.5}$ and network size $N = 2000$ as the contact network, and for the information network, which is generated by adding 800 extra random links in the contact network. a_{ij} and b_{ij} represent the adjacency matrices of the contact network and the information network, respectively. All simulation results are obtained by averaging 20 realizations.

We first compare the results from MMCA with Monte-Carlo simulation in Fig. 3 to check the effectiveness of our analysis based on MMCA, from Fig. 3, one can observe that the results based on the two approaches are in good agreement. So in the next figures, our main results are obtained from MMCA.

Then we investigate the effects of the two main parameters of the model— κ and σ on the epidemic threshold and the density of infected individuals. Here, we will present the results for $\gamma = 0$, meaning that $\beta^A = 0$ and $q^A = 0$. Obviously, once the value of γ is increased, the epidemic threshold is decreased and the density of infected individuals is enhanced correspondingly.

Fig. 4 plots the density of infection [ρ^I , see Fig. 4(a)] and aware individuals [ρ^A , see Fig. 4(b)] as a function of β for different values of κ , respectively. Observing the figure, one can see that though the larger value of κ can cause more individuals become aware and reduce the density of infection. However, one can find that the increasing of λ has no influence on the epidemic threshold. The result is remarkably different from many previous results which claim that the local information-based behavioral response in the *single-layer network* can alter the epidemic threshold. How to understand this nontrivial result? Since the UAU awareness dynamic is the same to the SIS epidemic process. When the epidemic has not broken out, the density of awareness in information network (*i.e.*, ρ^A) is only determined by the transmission rate of awareness, λ , recovery rate δ and the structure of information network, but is not related to the value of κ . Namely, near or below the epidemic threshold point, increasing the value of κ only means that the initial number of the aware individuals is increased, which cannot affect the density of aware individuals

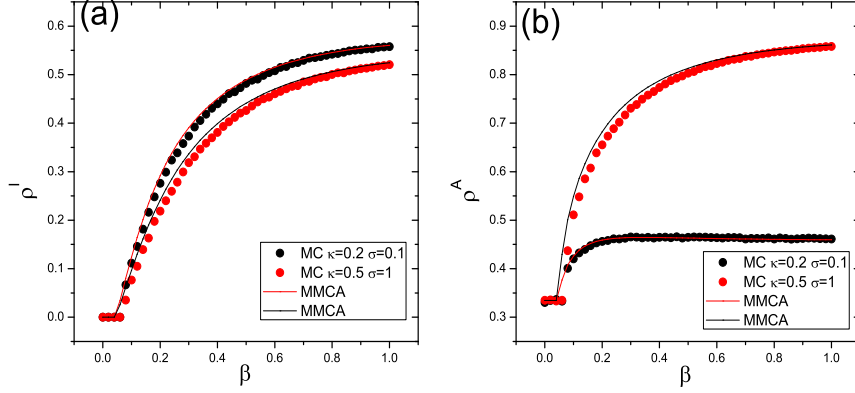


Fig. 3. Comparison of MMCA (solid line) with Monte Carlo simulation (dotted line) for $\lambda = 0.15$, $\gamma = 0$, $\delta = 0.6$ and $\mu = 0.4$. The fraction of infected (a) and aware (b) nodes as a function of the infectivity parameter β for two different conditions of the parameters κ and σ .

at stationary state. In this case, the value of p_i^A is independent of the value of κ or σ , which gives rise to the same value of ρ^A [see Fig. 4(b)]. Thus, according to Eqs. (13) and (15), the epidemic threshold β_c is invariable owing to the same value of ρ^A . One should note that, for the case of single-layer network, the local information-based behavioral response can *directly* reduce the transmission rate of epidemic, leading to the change of the epidemic threshold. For our model, the self-awareness behavior first diffuses through the information network, and then the epidemic process happens in contact network. Thus, the effect of the awareness behavior on the transmission rate is *indirect*.

The density of infected individuals and aware nodes as functions of β for different values of parameter σ are also shown in Fig. 5(a) and Fig. 5(b), respectively. One can see that, similar to Fig. 4, varying the value of σ has no effect on the epidemic threshold. However, differ to the above case, from Fig. 5(a) we find that the value of σ also has negligible effect on the density of infection, even in the extreme cases where infected unaware individuals remain unaware of its sickness ($\sigma = 0$) or certainly become aware of it ($\sigma = 1$). The result in Fig. 5 is in accord with the Fig. 3 in Ref. [24].

In order to systematically study the effects of κ and σ on the ρ^I , we further explore the full phase diagram ($\lambda - \beta$) in Fig. 6. Overall, we can see that ρ^I is not influenced by λ when β is smaller than the epidemic threshold, since epidemic will die out by itself. Once β overpasses the epidemic threshold, ρ^I decreases with λ for different values of κ or σ . More specifically, by comparing Fig. 6(a) with Fig. 6(b) (or comparing Fig. 6(c) with Fig. 6(d)), we can see that ρ^I is not remarkably influenced by the value of σ . Likewise, by comparing the Fig. 6(a) with Fig. 6(d) (or comparing Fig. 6(b) with Fig. 6(c)), ρ^I decreases with κ , especially for the large value of β .

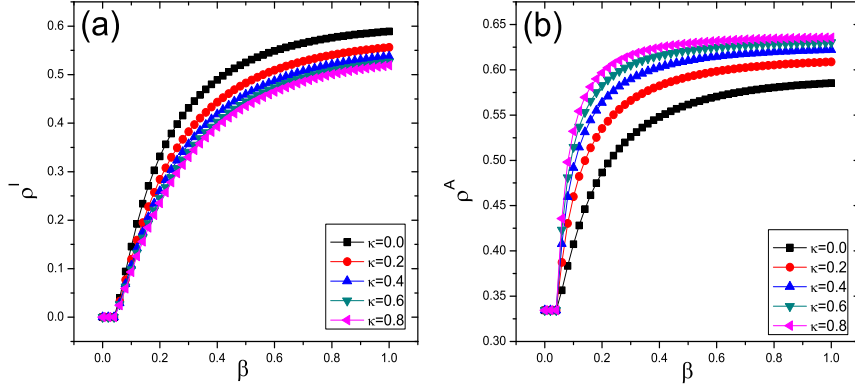


Fig. 4. Fraction of infected (a) and aware (b) nodes as a function of the infectivity parameter β for different values of the parameter κ . Here $\lambda = 0.15$, $\gamma = 0$, $\delta = 0.6$, $\mu = 0.4$ and $\sigma = 0.4$.

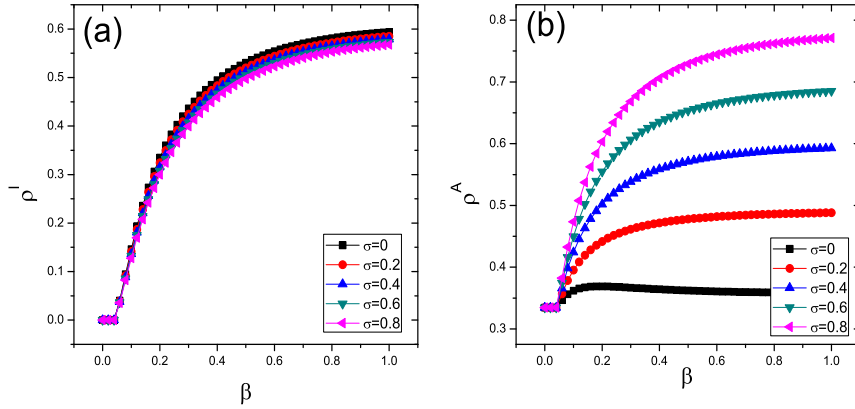


Fig. 5. Fraction of infected (a) and aware (b) nodes as a function of the infectivity parameter β for different values of the parameter σ . Here $\lambda = 0.15$, $\gamma = 0$, $\delta = 0.6$, $\mu = 0.4$ and $\kappa = 0.05$.

4 Conclusions and discussions

Recognizing that, on the one hand, outbreak of an epidemic through a physical-contact network can trigger the spreading of information awareness through other different channels, such as on-line social networks, mass media; on the other hand, an individual can not only be informed by other aware neighbors but also can become self-awareness once some friends in contact network are infected. By introducing the *self-awareness* mechanism for susceptible individuals, we have investigated interplay between the spreading of epidemic and the diffusion of awareness based on the framework of the multiplex networks. We mainly studied the two parameters κ and σ characterizing the self-awareness probability of susceptible individuals and infected individuals, respectively.

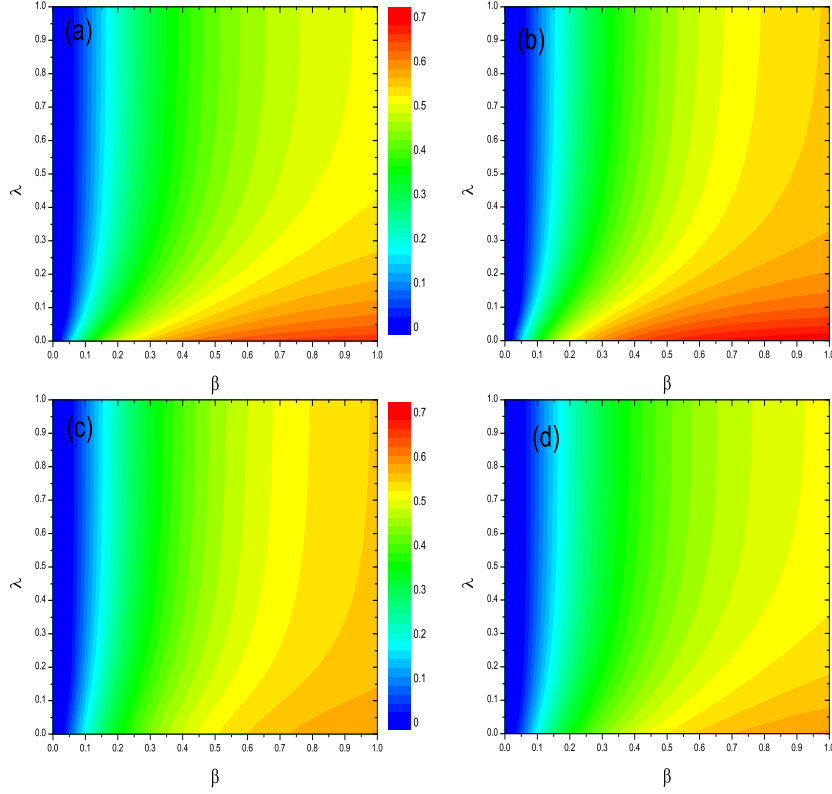


Fig. 6. The fraction ρ^I of infected individuals in the stationary state. Full phase diagram $\lambda - \beta$ for the same multiplex described before. Where $\gamma = 0$, $\lambda = 0.15$, $\delta = 0.6$ and $\mu = 0.4$. (a): $\kappa = 0$, $\sigma = 1$; (b): $\kappa = 0$, $\sigma = 0.2$; (c): $\kappa = 0.2$, $\sigma = 0.2$; (d): $\kappa = 0.2$, $\sigma = 1$.

Analysis based on the Markov chain approach as well as the extensive computations reveal that the density of infection can be reduced once the two parameters are increased, however, we found that the impact of self-awareness behavior for susceptible individuals on inhibiting the spreading of epidemic is much better than the self-awareness of the infected individuals, since self-awareness from susceptible individuals can directly reduce their probabilities of being infected. Meanwhile, we found that the self-awareness behavior cannot alter the epidemic threshold no matter of the local or global information, which are in stark contrast with the results obtained from the single-layer networks.

The challenges of studying the intricate interplay between social and biological contagions in human populations are generating interesting science. In this work, we consider the effects of the *self-awareness* behavior based on the

multiplex networks on the density of infection and the epidemic threshold, our result implies that the conclusions obtained from single-layer networks may need to be re-examined when they are extended to multiplex networks.

5 Acknowledgments

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6 Appendix: Global information-based awareness

In recent work, Granell *et al.* considered the effect of the mass media on the epidemic process and awareness diffusion [24]. In the model, each individual becomes aware with probability m by assuming that they are informed by a broadcast or mass media. Thus, it can be regarded as a global information-based awareness. One questionable point is that the probability of being awareness m is irrelevant to the density of infection. As a result, even the epidemic is almost eliminated, individuals also have the fix probability of being aware. In reality, becoming awareness often means that individuals need to take some protective measures, such as, washing hands, wearing masks or reducing outgoings. These measures indicate certain inconveniences or some cost [27,28]. Thus, a more realistic situation is that the probability of being awareness should be related to the density of infection. To mimic this case, here we assume the probability of aware from global information is given as: $mI(t)$ with $I(t)$ is the density of infection at time t , which indicates that the probability of being awareness adaptively varies with the density of infection.

For this case, we only need to slightly change the local model described in subsection 3.1. We only need to change $\gamma_i(t)$ and $\theta_i(t)$ as follows:

$$\gamma_i(t) = \prod_j (1 - b_{ji} p_j^A(t) \lambda) (1 - m p^I(t)), \quad (16)$$

$$\theta_i(t) = \prod_j (1 - b_{ji} p_j^A(t) \lambda) (1 - a_{ji} p_j^I(t) \kappa) (1 - m p^I(t)) \quad (17)$$

Similarly to the above analysis, we can get that the epidemic threshold is still determined by Eq. (15), *i.e.*, the epidemic threshold is also independent of the value of m , which is different from the result in Ref. [24]. The result is verified by Fig. 7 for different values of γ .

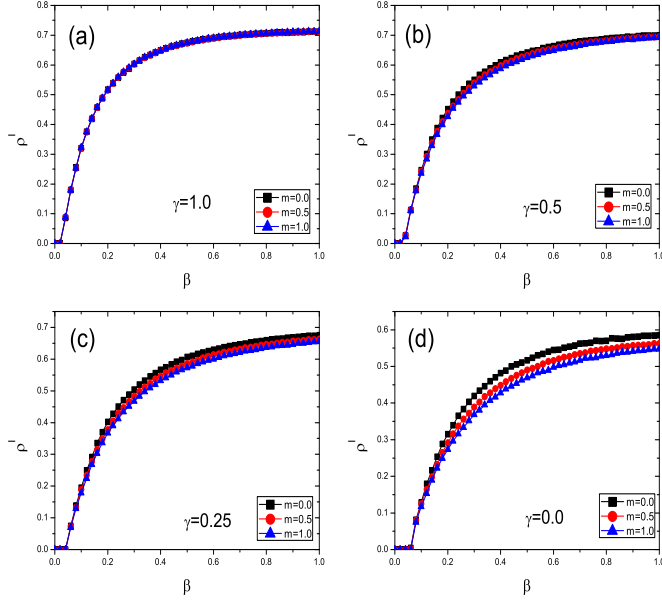


Fig. 7. The density of infected individuals as a function of β for different values of m . Here $\lambda = 0.3$, $\sigma = 0.5$, $\delta = 0.6$, $\mu = 0.4$ and $\kappa = 0.0$. (a): $\gamma = 1$; (b): $\gamma = 0.5$; (c): $\gamma = 0.25$; (d): $\gamma = 0$.

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